Environmental Exposure Impacts Upon Human Health

Introduction

Californians are concerned about the harmful effects of environmental pollutants on their health. Sharing this recognition that exposures to environmental contaminants have the potential to adversely impact human health, Cal/EPA and the Department of Health Services seek to reduce or eliminate potentially harmful exposures to hazardous chemicals in the environment.

It is not always easy to determine when environmental pollutants produce disease. Disease occurrence is a product of many factors that influence progression from wellness to disease, beginning with imperceptible or subtle changes in normal biochemical activity, followed by measurable impairments in body function, the obvious appearance of disease, and ultimately death. Specific susceptibilities to particular illnesses may be inherited or acquired. Some individuals may be predisposed to specific diseases because of certain genes inherited from their parents. For example, over 40 percent of all individuals with retinoblastomas (a rare tumor affecting the retina) have inherited the susceptibility for that cancer (Paulino, 1998). Colorectal, breast, and ovarian cancers, some forms of acute and chronic leukemia, and other forms of cancer have been shown to run in families due to hereditary influences. Disease susceptibility is also strongly influenced by aging, and by many factors including infections, exposure to hazardous environmental chemicals, and certain lifestyle behaviors. Our understanding of cancer risk factors remains incomplete, however, lifestyle factors alone, particularly smoking, diet, and lack of exercise may contribute to a majority of known cancer risks (Harvard Center for Cancer Prevention, 1996).

Conversely, certain beneficial factors can promote health, counteracting the influences of detrimental factors. Disease progression can be slowed by healthy lifestyle choices such as good nutrition, routine exercise, avoidance of tobacco use, positive social environment, and medical treatment. These factors can reverse or delay the disease process, improving the quality of, and prolonging life. Largely due to sanitary measures, the adoption of healthier lifestyles and improvements in the quality of medical care, there have been steady declines in infant mortality rates and increases in life expectancy (see *Infant Mortality* and *Life Expectancy* in the "Background Indicators" section).

In some instances, the predominant factor in human health can be exposure to environmental pollutants. A number of tragic examples clearly demonstrate a causal relationship between environmental pollutants and acute or chronic disease. Severe

Indicators of Environmental Exposure Impacts on Human Health

Human body concentrations of toxic chemicals

Surveillance of persistent organic pollutants in body tissues and fluids

Concentrations of persistent organic pollutants in human milk (Type III)

Lead in children and adults

Elevated blood lead levels in children (Type II)

Mercury in children and adults

Mercury levels in blood and other tissues (Type III)

birth defects occurred among a large number of infants born to women who consumed seafood contaminated with methylmercury caught from Minamata Bay, Japan. In Libby, Montana, many workers, their families, and local residents developed asbestosis and mesothelioma following exposures to asbestos and asbestos-like minerals from vermiculite mining activities. Finally, the widespread use of lead-based products, including gasoline and paint, until the 1970s caused thousands of children to suffer from severe lead poisoning, while many more suffered from subtle lifelong neurotoxic effects before these products were banned. Although much of the toxic effects of lead had been known for centuries, the public was largely uninformed about the potential devastating effects that these lead-containing products would have on children.

While large or unusual exposures to environmental contaminants can result in detectable increases in the numbers of disease cases in a population, disease from smaller or limited exposures are often not detectable. Sometimes environmentally-caused illnesses are subtle, or occur many years after the exposure. In addition, the health influences of factors other than environmental exposures (including genetics, diet, smoking and other lifestyle choices), or illnesses unrelated to the environmental exposure make it hard to distinguish to what extent environmental pollutants have contributed toward observed diseases.

How does Cal/EPA protect public health?

Protecting the public health from exposures to harmful environmental contaminants involves a process consisting of two phases: risk assessment and risk management. In the risk assessment phase, the likelihood of adverse health effects resulting from human exposures to environmental contaminants is evaluated. In the risk management phase, regulatory standards or criteria are developed and implemented to manage or eliminate harmful exposures to hazardous chemicals. For example, Cal/EPA's Office of Environmental Health Hazard Assessment (OEHHA) conducts risk assessments to develop human health protective guidelines for contaminants in drinking water (called public health goals); the Department of Health Services then considers these guidelines in risk management to promulgate regulatory standards (called maximum contaminant levels) to ensure the safety of drinking water.

Human health protection is often the underlying basis for many environmental regulations. Over the years, these regulations have led to significant reductions in the levels of contaminants in the environment as well as the prevention of further contamination. Table I is a list of the environmental indicators discussed in other sections of this chapter, which reflect and track the extent to which regulatory standards are met. These indicators provide an indirect measure of how well the public is protected from environmental contaminants.

What indicators are presented in this section?

The issues and indicators in this section focus on characterizing the impacts of environmental contaminants on human health. The discussions provide an overview of the state of knowledge regarding the association among exposures to environmental pollutants, concentrations of contaminants in the body (also known as "body burdens"), and diseases associated with exposures to environmental contaminants.

Tracking chemical body burdens has been found to aid in determining which individuals are at risk for disease, preventing the occurrence of disease, and determining the sources of exposure. For example, the detection and monitoring of blood lead levels in children is used to identify children in need of treatment to prevent lead poisoning. These data are valuable in developing effective measures to identify and reduce sources of lead in the environment.

This section broadly discusses several disease categories as issues of general interest associated with environmental contaminants. These categories are cancer, respiratory disease, and reproductive effects. Because of the lack of data as well as the complexity of the interactions among the various factors that produce disease, no indicators are proposed for this category. Rather, continuing or enhancing disease surveillance will be useful in understanding trends and providing information on causation.

Surveillance systems that track body burdens of toxic contaminants of concern and the incidence of environmentally-related diseases represent effective tools for understanding how body burdens or human diseases are influenced by environmental exposures. Based on information provided by surveillance systems, Cal/EPA can better formulate informed responses to environmental challenges.

Environmental Indicators Related to Public Health Protection
Air Quality
Days with unhealthy levels of ozone pollution
Peak 1-hour ozone concentration
Exposure to unhealthy ozone levels in the South Coast air basin
Emissions of ozone precursors (VOC + NOx)
Days with unhealthy levels of inhalable particulate matter (PM10)
Peak 24-hour PM10 concentration
Annual PM10 concentration
Total primary and precursor PM10 emissions
Days with unhealthy levels of carbon monoxide
Peak 8-hour carbon monoxide concentration
Carbon monoxide emissions
Total emissions of toxic air contaminants (TACs)
Community-based cancer risk from exposure to TACs
Cumulative exposure to TACs that may pose chronic or acute health risks
Indoor exposure to formaldehyde
Household exposure of children to environmental tobacco smoke (ETS)
Water
Drinking water supplies exceeding maximum contaminant levels
Total open leaking underground fuel tanks (LUFTs) sites
Groundwater contaminant plumes- Extent
Contaminant release sites
Coastal beach availability- Extent of coastal beaches posted or closed
Aquatic life and swimming uses assessed in 2000
Fish advisories - coastal waters
Fish advisories – inland waters
Bacterial concentrations in commercial shellfish growing waters
Land, Waste and Materials Management
Soil cleanup
Contaminated sites
Pesticides
Number of detections of pesticides identified as toxic air contaminants and the percent that exceeds numerical health standards each year
Area with pesticides detected in well water
Simazine and breakdown products in a monitoring network of 70 wells in Fresno and Tulare Counties
Pesticide detections in surface water and the percent that exceeds water quality standards
Percent of produce with illegal pesticide residues
Pesticide use volumes and acres treated, by toxicological and environmental impact categories
Number of growers adopting reduced-risk pest management systems and the percent reduction in use of high risk-pesticides (based on Alliance grant targets)
Number of reported occupational illnesses and injuries associated with pesticide exposure
Transboundary
Air pollutants at the California/Baja California, Mexico Border

Issue 1: Human Body Concentrations of Toxic Chemicals

Certain toxic chemicals, although present in low concentrations in the environment, may accumulate in human tissue because they resist environmental or biological degradation. These chemicals may pose an increased health risk as their tissue concentrations increase. For these chemicals, efforts are focused on avoiding and reducing exposures as much as technically feasible, even when adverse health effects are unknown. However, it is not always possible to know where exposures are coming from. Tracking the concentrations of these chemicals in the body (i.e., "body burden" levels) by a sustained, routine biological tissue monitoring system may yield valuable information on potential sources of environmental contaminants.

Indicator

Concentrations of persistent organic pollutants in human milk (Type III)

Sub-issue 1.1: Surveillance of persistent organic pollutants in body tissues and fluids

The persistent organic pollutants (POPs) are a large class of compounds that include polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), polychlorinated dioxins and furans, and certain pesticides, such as DDT, aldrin or dieldrin. These chemicals can enter the body through many exposure pathways from environmental media containing these chemicals, including air, food and water. Once in the body, these compounds tend to reside in fatty tissues where they persist until they are mobilized by various conditions. During pregnancy, these chemicals are carried from body fat by the blood through the placenta to the fetus. They can be ingested in breast milk by a nursing child or mobilized during weight loss.

Exposures to these compounds are of concern because they are known to affect certain hormonal pathways and some are associated with detrimental health effects. For example, some of the POPs are either known or suspected endocrine disruptors or carcinogens. No routine, ongoing surveillance system exists to monitor POPs in human tissues. The establishment of such a surveillance system for breast milk, fat tissues, and other systems would be beneficial (USEPA, 1998).

Indicator

Elevated blood levels in children (Type II)

Sub-issue 1.2: Lead in children and adults

Lead is a neurotoxin that impairs cognitive function and physical development, particularly in young children. For more than a decade, inorganic lead exposure to young children has been the major pediatric environmental health concern, particularly of poor children living in old, substandard housing where lead-based paint is often in a deteriorating condition. Blood lead levels greater than 10 micrograms per deciliter (µg/dL) in children are associated with lower intelligence and reading ability, learning disabilities, impaired hearing, reduced attention spans, and many other cognitive and physical problems. Subtle neurological and biochemical effects at levels lower than 10 µg/dL have been reported in the scientific literature. At this point, no threshold blood level concentration, below which no effect is expected to occur, has been determined. Adults exposed to high levels of lead, typically through occupational exposures, may have kidney toxicity, anemia, and nervous system disorders.

Historically, millions of tons of lead have been used in a wide array of consumer products in the United States. Because lead is an element, it will not degrade and will remain in the environment where it is released. With interventions, such as elimination of lead in gasoline and paint, excellent progress has been made in reducing the average blood lead levels in the overall population. Data from the Centers for Disease Control (CDC)'s Third National Health and Nutrition Examination Survey, Phase 2 (1991-1994) and the 1999 update showed that average blood lead levels decreased by approximately 80 percent since the 1970's (CDC, 2000). During the years 1976-1980 to the period of 1988-1991, the geometric mean of blood lead values declined from 12.8 µg/dL to 2.9 µg/dL. The levels further declined during the period of 1991-1994 to a geometric mean of 2.3 µg/dL. This survey also identified elevated blood lead levels in low-income children, children in urban areas and those living in older housing. Lead in deteriorating paint in housing constructed prior to the banning of lead in paint, soils contaminated with lead paint or deposits of lead from past gasoline emissions, and other sources continue to present possible sources of lead exposures. As older housing is renovated, lead exposure from this source should decrease. Nationally, children's blood lead levels have continued to decline in the 1990s (CDC, 2000).

Each year dozens of adults are poisoned with lead, primarily from occupational exposures. This is a particular problem because of potential adverse reproductive effects in males and females, as well as a substantial hazard to the developing fetus. Blood lead levels of adult workers are reported only when they exceed 25 µg/dL.

Sub-issue 1.3: Mercury in children and adults

Mercury and its compounds in the environment are derived from both natural sources and from human activity. In California, large amounts of mercury were released during mining for either mercury or gold ore into streams and lakes over the last two centuries, although relatively small concentrations of mercury were always present in the waters. Mercury poses a particular public health problem when it is discharged into aquatic bodies where the inorganic mercury is converted by microorganisms to the much more toxic form, methylmercury. When methylmercury contaminates the food chain, it biomagnifies in some aquatic organisms including fish, thereby posing a potential health hazard when ingested by humans.

Indicator

Mercury levels in blood and other tissues (Type III)

For the general population, the principal exposure pathways for mercury are inhalation of airborne mercury from dental amalgams and ingestion of fish (fresh water and marine) and other seafood containing methylmercury.
At higher exposure levels in adults, mercury may adversely affect the kidneys

and the immune, neurological, respiratory, cardiovascular, gastrointestinal, and hematological systems. The developing nervous system is especially sensitive to the toxic effects of low-level mercury exposure. Methyl mercury will cause birth defects or fetal death when pregnant women ingest sufficient quantities of methyl mercury (USEPA, 2001).

Issue 2: Environmentally Associated Diseases and Conditions

Environmental exposures to chemicals have been associated with certain human diseases. These effects have been found by observation or surveillance of unusual patterns, including new occurrences, of diseases. The effects of environmental pollutants may not always be detected by surveillance system. Nevertheless in the past, such surveillance had led to effective efforts to protect against harmful exposures to environmental pollutants, and to an understanding of the relationship between exposures to environmental chemicals and disease.

Sub-issue 2.1. Cancer

Cancer is a group of diseases which is recognized to be the second leading cause of death for Californians (see “Background Indicators” section). Generally, it is recognized that the exposure to environmental pollutants contributes less to the overall population cancer risk than other factors (Melse and deHollander; 2001; Doll, 1999). Smoking, diet, inactivity, and obesity have been identified as major cancer risk factors, and may account for about two-thirds of all cancer deaths (Harvard Center for Cancer Prevention, 1996). In addition to these major factors, other known contributing factors include alcohol consumption, viruses, genetics, radiation, and prescription drugs. Given the multiple factors that contribute to the risk of cancer, the long latency times between exposure to the onset of cancer, and the low levels at which chemicals usually occur in the ambient environment, associating cancer with specific environmental exposures becomes difficult.

Cancer is predominantly an adult disease increasing in incidence with age. Childhood cancers are generally rare, occurring at a rate of 15.2 cases per 100,000 U.S. children for 1998. By contrast, overall cancer incidence rate for all ages in the U.S. is 400.5 cases per 100,000. (SEER, 2001). For the past 25 years, the national childhood cancer incidence rate has remained generally stable. From the period of 1988 to 1994, the childhood cancer incidence rates in California are similar to the national rates (CCR, 1999). Childhood cancer is a concern because of the severity of the illness, the potential for delayed development, and premature deaths. Fortunately, successful treatment of childhood cancers has dramatically decreased mortality, to about half the death rate since 1973 (CCR, 1999).

As with adult cancer cases, childhood cancers can be the result of many factors. According to the 1999 report, "National Cancer Institute Research on Causes of Cancers in Children" (NCI, 1999) there are very few known causes of cancer in children. Those that have been identified to date include Down's syndrome, other specific chromosomal and genetic abnormalities, and treatment with radiation or chemotherapy. These causes are thought to contribute to only a small proportion of the cancers in children. Some research in this area suggests that exposure to certain environmental toxicants early in life may be linked to the development of certain childhood cancers.

Identifying and examining potential links between environmental agents and cancer are ongoing. Evidence of the link between environmental agents and cancer generally requires exposures at levels many times higher than those expected to occur in the ambient environment, such as those observed in occupational settings or from certain therapeutic drugs used to treat diseases, including cancer itself. Complicating this picture is the fact that people are exposed to many other substances that may affect the risk of cancer. The continuing efforts by the California Cancer Registry and California Department of Health Services to monitor detailed data on cancer incidence, mortality, and survival will contribute to the understanding of the causes and mechanisms of cancer (see www.ccrcal.org).

Sub-issue 2.2: Respiratory disease

Environmental pollutants are associated with increased acute respiratory disease morbidity; aggravation of asthma; increased prevalence of respiratory symptoms in children including prolonging infectious episodes; and decreasing lung function in children.

In particular, asthma is one of the most serious chronic respiratory diseases both in this country and around the world. For many years, the number of new cases has been increasing, particularly among children and adolescents (see asthma indicators in the "Background Indicators" section). It has been suggested that environmental factors, including exposure to certain air pollutants, are contributors to these increases. Yet, trends in the ambient levels of the most troublesome air pollutants in California, ozone and particulate matter, have been proceeding in the opposite direction or have remained stable, relative to the trends reported for asthma. Ozone levels in large California cities have been declining for many years, while particulate matter levels have had moderate declines or have been relatively constant. To begin to understand this dilemma, there are two fundamental, but separate, issues with regard to asthma and environmental factors that need to be considered. These are: (1) factors leading to the development or onset of asthma, particularly in children; and, (2) causes of aggravation, or exacerbation, of pre-existing asthma symptoms.

The environmental factors involved in the onset of asthma are complex and incompletely understood. However, many researchers suspect that the rate of asthma development among the population is increasing, resulting in more asthmatic attacks due to poor air quality and other factors, even though overall levels of air pollutants have declined. Ozone exposure has been implicated in the development of asthma, but it has become apparent that other more important factors may contribute to the onset of this disease. Such factors include genetics, exposure to allergens (such as those from dust mites and cockroaches) and indoor air quality pollution (such as respiratory viruses and environmental tobacco smoke).

In contrast, a number of air pollution studies in California (Koren, 1995) and other states have noted increased asthma hospital admissions or emergency visits associated with high levels of outdoor air pollutants in the regional air. California will soon require emergency room visits as reportable data (OSHPD, 2001), providing a possible means for tracking emergency room asthma visits. However, due to considerable changes in health care management, simply tracking asthma-related emergency room visits would not be a reliable indicator of poor air quality days. Successful asthma management has led to the decrease in the number of acute attacks requiring emergency room visits. Furthermore, very specialized expertise is needed to assess asthma-related emergency room visits and to track the history of the patient to identify exposures to other pollutants and allergens unrelated to outdoor air pollutants.

Sub-issue 2.3: Reproductive and developmental health effects

Various factors have long been known to influence adverse reproductive and developmental health outcomes apart from heredity. Since the 1940s, it has been known that mothers infected with rubella had a higher risk of losing their offspring, or of having their offspring affected by blindness and deafness. Later the drug thalidomide was shown to cause an increase in malformed fetuses among pregnant women taking that drug to reduce nausea. Recently, it has been shown that folic acid taken as a dietary supplement by pregnant women can reduce the incidence of spinal cord and brain birth defects.

In addition to birth defects, chemicals such as mercury can also cause fetal loss or miscarriages from prenatal exposure. To complicate the picture, there are a great variety of defects and disorders that are not obvious or diagnosed until later in life, including learning deficiencies or neurological impairments.

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As with cancer and respiratory disease, development and reproductive effects are influenced by many factors, and the degree to which environmental pollutants contribute to these outcomes is not thoroughly understood. The effects of chemicals on the overall rates of birth defects or reproductive outcomes are difficult to address, unless the effect is a rare defect and highly associated with the agent, and is rare. For example, thalidomide was identified as a causative agent when there was a high incidence of a very rare birth defect. Surveillance of birth defects (conducted by the California Birth Defects Monitoring Program, www.cbdmp.org) will play an important role in understanding the causes of birth defects and reproductive effects.

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Type II

Elevated Blood Lead Levels in Children

Childhood lead poisoning is the most common environmental health problem for children in the United States. It is usually silent, detected only when a child has a positive blood lead screening test. Left undetected, lead exposure causes lowered IQ, learning disabilities, attention deficit disorder, and other problems associated with the nervous system. In the long term, elevated blood lead levels may result in an increased likelihood for school failure and lower lifetime earnings potential.

Most lead exposure results from the presence of lead-based paint in older residential housing. Children are exposed when the paint is peeling, or is disturbed during renovations. Dust and soil in and around older housing can also be contaminated. Therefore, children in lower income families are at a higher risk of lead poisoning because they are more likely to live or spend time in old substandard housing. Not as commonly, children may be exposed to other sources of lead, including the use of certain ethnic remedies and cosmetics, imported lead-contaminated food products, and traditional ceramic cooking ware.

The federal Centers for Disease Control and Prevention (CDC) currently defines “elevated blood lead levels” as 10 micrograms per deciliter (µg/dL) or higher. New California regulations require clinicians to screen children for elevated blood lead levels at 12 and 24 months of age if the child receives assistance from a publicly funded program such as Medi-Cal, the Women, Infants and Children Program (WIC), or the Child Health and Disability Prevention Program, or if the child lives in a house built prior to 1978 that has chipped or peeling paint or that has recently been renovated. At present, only two laboratories report all childhood blood lead levels to the Childhood Lead Poisoning Prevention Branch of the Department of Health Services (DHS). Several other large laboratories around the state will begin electronic reporting of all blood lead levels over the coming year. As a result, data from more sites are expected to be available to estimate the prevalence of elevated blood lead levels among children in other parts of California.

As California’s aging housing stock containing lead-based paint is remediated, it is anticipated that there will be a continued trend in decreasing blood lead concentrations.

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Concentrations of Persistent Organic Pollutants in Human Milk

Type III

The persistent organic pollutants (POPs) are an important class of compounds, which include polychlorinated biphenyls, polybrominated diphenyl ethers, polychlorinated dioxins and furans, and certain pesticides. These compounds were used in a variety of products and have been distributed worldwide. POPs resist environmental degradation and persist in the human body. They have known biologic activity; that is, they react to various sites on cells, and alter cellular function. Certain POPs have also been associated with a number of detrimental health effects, from altered sex ratio to cancer. Some are also known to act as endocrine disruptors, which means they affect hormone activity; this may account for some of the associated health effects.

POPs generally reside or accumulate in high-fat containing tissues. Lactating mothers utilize their fat stores to produce breast milk, and in so doing mobilize the POPs stored in fat as contaminants into the milk. Therefore, human milk is a simple, non-invasive means to monitor POP body burdens.

Although isolated studies conducted on human breast milk and human fat in California indicate that POPs are present, no consistent monitoring is being conducted at this time. Thus a study would need to be designed and initiated to address this issue.

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Mercury Levels in Blood and Other Tissues

Type III

Californians consuming sport fish from lakes and estuaries, containing large amounts of mercury in the sediment, are at risk for having higher body burdens of mercury. Mercury in the environment comes from both natural and man-made sources, and is available in several different forms. These forms interconvert among each other depending upon chemical and physical conditions. Of particular concern is the conversion by aquatic microorganisms of inorganic mercury to methylmercury, a particularly toxic form of mercury. The mercury is transferred from microorganisms to fish, and then to humans consuming these fish. While the brain and the kidneys are the primary targets of mercury toxicity, the developing nervous system in children is especially sensitive to the toxic effects of low-level methylmercury exposure. Thus, exposures to mercury is of great concern for women of childbearing age.

Recent preliminary estimates of blood and hair mercury levels come from the 1999 National Health and Nutrition Examination Survey (NHANES, see www.cdc.gov/nchs/nhanes.htm). NHANES is a continuous survey of the health and nutritional status of the U.S. civilian, non-institutionalized population. A summary of the most recent national data for mercury in blood from NHANES is presented below:

Total blood mercury concentrations (in µg/L) for U.S. children and women

	Sample size	Geometric Mean (95% Confidence Interval)	Selected Percentiles (95% Confidence Interval) 10th	25th	50th	75th	90th
Children-1-5 years	248	0.3 (0.2-0.4)	<LOD*	<LOD	0.2 (0.2-0.3)	0.5 (0.4-0.8)	1.4** (0.7-4.8)
Females-16-49 years	679	1.2 (0.9-1.6)	0.2 (0.1-0.3)	0.5 (0.4-0.7)	1.2 (0.8-1.6)	2.7 (1.8-4.5)	6.2 (4.7-7.9)
Source: National Health and Nutrition Examination Survey, 1999							

< LOD means below the limit of detection of the analytical method.
* less than the limit of detection of 0.1µg/L blood.
** Estimate meets minimum standards of reliability, but should be interpreted with caution.
Numbers in parentheses are 95% confidence intervals.

The National Research Council (NRC) completed a toxicologic review of mercury and computed a benchmark dose (BMD) for methylmercury exposure to the fetus associated with an increase in abnormal scores on cognitive function tests in children. The lower 95 percent confidence bound of the BMD was 58 µg/L. The 90th percentiles of mercury levels in children 1 through 5 years old and women of childbearing age are below this level. Approximately 10 percent of women have mercury levels within one-tenth of this level. This study suggests that mercury levels in young children and women of childbearing age are currently below those considered hazardous.

Further monitoring will provide trends regarding the levels of mercury in average Americans. Although national data are and will be available for mercury, California-specific information is not. Mercury is a major concern for those who might consume sport fish derived from California lakes and estuaries. Therefore, in order to address the issue of what are the mercury body burdens for Californians, specific surveillance data need to be obtained.

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